Recombinant Human
Lymphotoxin βR/TNFRSF3 Fc Chimera
Catalog Number: 629-LR

DESCRIPTION

Source
Mouse myeloma cell line, NS0-derived

Human LTβR (Ser28-Met227) & (Gln31-Met227) 
Accession # NP_002333

DIEGRMD

Human IgG1, (Pro100-Lys330)

6-His tag

N-terminal Sequence
Ser28 & No results obtained: Gln31 predicted

Analysis

Structure / Form
Disulfide-linked homodimer

Predicted Molecular Mass
49.6 kDa (monomer)

SPECIFICATIONS

SDS-PAGE
70 kDa, reducing conditions

Activity
Immovilized LT βR/Fc Chimera has been used for affinity purification of Lymphotoxin α1/β2 heterotrimer.

Endotoxin Level
<0.10 EU per 1 µg of the protein by the LAL method.

Purity
>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation
Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution
Reconstitute at 100 µg/mL in sterile PBS.

Shipping
The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage
Use a manual defrost freezer and avoid repeated freeze-thaw cycles.
- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Lymphotoxin beta receptor (LTβR), previously called TNF RIII or TNF R-related protein (TNF Rrp), is a type I transmembrane glycoprotein within the TNF receptor superfamily, designated TNFRSF3 (1-3). Human LTβR cDNA encodes 435 amino acids (aa) including a 30 aa signal peptide, a 197 aa extracellular domain (ECD), a 21 aa transmembrane domain, and a 187 aa cytoplasmic domain. The ECD contains four cysteine-rich motifs characteristic of the TNF receptor superfamily (1, 2). Within the ECD, human LTβR shares 67-74% aa sequence identity with mouse, rat, canine, porcine, equine and bovine LTβR. Soluble LTβR can be formed by proteolytic cleavage of the ECD, and is an inhibitor of transmembrane LTβR, which inhibits autommunity (3-6). Potential human isoforms include a 416 aa form with an alternate N-terminal signal sequence, and a 328 aa form that begins at aa 108 (7). LTβR is expressed by visceral, lymphoid, and other stroma, epithelia and myeloid cells, but not lymphocytes (2, 4). LTβR ligands include homotrimers of LIGHT (TNFSF14; also a ligand for HVEM) and the heterotrimeric lymphotoxin LTα1β2 (3, 4, 6). Depending on the cell type and expression of TRAF3, activation of LTβR has been shown to induce canonical (IKK/RelA; pro-inflammatory) or alternative (NIK/RelB; lymphoid organogenic) NFκB activation (6, 8). LTβR is expressed on mesenchymal stromal organizing cells that give rise to stroma of primary (thymus), secondary (tonsils, lymph nodes and Peyers patches) and tertiary (ectopic inflammatory) lymphoid structures (3-5, 9-11). Secondary immune tissues are absent in LTβR-deficient mice (3-5). LTβR engagement induces production of IL-7, RANK, TRANCE/RANK L, VEGF-C, adhesion molecules such as VCAM-1, ICAM-1 and MadCAM, and chemokines such as CXCL13, CCL19 and CCL21 (3, 9-11). LTβR is expressed by hepatocytes, is up-regulated in regeneration, hepatitis and hepatocellular carcinoma, and influences lipid metabolism and atherosclerosis (4, 6, 12). It regulates cell growth and can initiate inflammation-related carcinogenesis (6, 12).

References:
7. Entrez accession # BAH11468 and BAG53051.

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